

Recent Developments in Research of Trauma and Personality Disorders

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Although the association between trauma and personality disorders, particularly borderline personality disorder (BPD), has been well established, the etiologic role of trauma in the development of personality disorders has been a topic of debate. Numerous mediation models have been put forth to explain how trauma can serve as a risk factor for the subsequent development of BPD. The symptomatic overlap between the proposed complex post-traumatic stress disorder diagnosis and BPD has fueled research efforts aimed at determining whether these are distinct disorders or should both be considered as trauma spectrum disorders. Treatment implications of this diagnostic differentiation are discussed.

Introduction

Since Herman *et al.*'s [1] pivotal study in which 81% of borderline personality disorder (BPD) subjects reported a history of trauma, numerous research studies have consistently found a greater incidence of childhood trauma in the form of sexual and physical abuse among BPD subjects, when compared with other psychiatric controls groups [2–4]. It is generally estimated that 55% to 80% of BPD patients have experienced some severe childhood trauma, and at least one third of BPD patients meet strict criteria for comorbid post-traumatic stress disorder (PTSD) [3]. This compelling association has led some to conclude that childhood trauma is one of the principal etiologic factors in severe personality disorders [1,5]. However, because many individuals with BPD are without trauma histories, and conversely, many with histories of severe trauma do not have a personality disorder diagnosis, a more cautious interpretation of these collective findings has been advised [6]. The ongoing debate over the etiologic role of childhood trauma in personality disorders has generated much research aimed at understanding the specific parameters of trauma that may contribute to personality disorder symptomatology.

Specific Traumas and Personality Disorder Profiles

Most of the literature on the association between trauma and personality disorder focuses on childhood sexual abuse and BPD. As reviewed by Gunderson and Sabo [2], in the 5 years since the Herman *et al.* [1] study, there were at least 11 studies involving over 600 patients in aggregate, which found a greater incidence of childhood trauma in BPD patients compared with other psychiatric populations. Recently, a study involving 112 patients who met criteria for a DSM-III-R (*Diagnostic and Statistical Manual of Mental Disorders*, revised 3rd edition) personality disorder found that trauma in the form of sexual abuse was more common in BPD compared with other personality disorder patients [7]. However, results failed to find differences between BPD and other personality disorders with regard to other types of trauma (*eg*, crime events, general disaster events, or witness of traumatic events), and whether traumatic events occurred in childhood or adolescence. A specific association between BPD and sexual abuse, arguably distinct from other types of trauma in that it is often ongoing and involves a high degree of personal violation, has been consistently reported in much of the clinical and empiric literature.

Combat-related trauma has also been linked to a higher incidence of Axis II disorders, albeit with less specificity. When assessed by structured interview, combat veterans were found to have high rates of borderline, avoidant, and obsessive-compulsive personality disorders [8]. Studies using self-report measures found high rates of borderline, avoidant, passive-aggressive, antisocial, schizotypal, and schizoid personality disorders among combat veterans with PTSD [9–11]. There have been inconsistent associations reported between exposure to combat trauma and high incidence of antisocial personality disorder [12].

A recent study examined personality disorder features among outpatient combat veterans, inpatients with a history of sexual abuse, and outpatients with a history of sexual abuse, and compared them with other psychiatric samples on personality disorder features [13•]. A clear pattern of higher correlations within the three trauma samples than between trauma versus nontrauma samples emerged, suggesting that some specificity of personality disorder features is shared by various trauma samples. The personality disorders most frequently represented across all trauma samples were paranoid, borderline, schizotypal,

and self-defeating. The combat veterans sample also exhibited relatively high elevations on obsessive-compulsive, schizoid, and passive-aggressive personality disorders. Although both childhood sexual abuse samples had near identical profiles, one striking exception was the difference in rates of BPD. Among the inpatient sample, over 90% met threshold criteria for BPD, in contrast to approximately 50% of the outpatient sexual abuse sample.

One study that examined relationships between types of childhood maltreatment and personality disorders in a substance abuse population supports the need to differentiate between types of childhood trauma in considering their possible effects on personality disturbance [14••]. Their results indicated that physical abuse and neglect were related to antisocial and sadistic personality traits, emotional neglect was related to traits of schizoid personality disorder, emotional abuse was a broad risk factor for personality disorders in clusters A, B, and C, but sexual abuse, which had been predicted to be related to BPD, was found to be unrelated to any particular personality disorder cluster. However, the sample was comprised of primarily male substance abusers. In spite of this, the cumulative evidence suggests that although various trauma groups share a degree of specificity in features and commonality of symptoms, there does appear to be some support for specific associations between types of trauma and personality disturbance.

Interface Between Trauma and Personality Disorder: Diagnostic Implications

The observed overlap between trauma and BPD, have led to the suggestion that the latter be considered a trauma spectrum disorder [1]. Among those suffering from PTSD, an identifiable subset experience persistent and chronic symptoms, and appear to have integrated aspects of the trauma reaction with their personality. This clinical observation has led to the conceptualization of the complex PTSD (CP) diagnosis, also referred to as *Disorders of Extreme Stress Not Otherwise Specified* (DESNOS). Features of this proposed trauma spectrum diagnosis include affect dysregulation, risky and self-destructive behaviors, identity disturbance, dissociative experiences, feelings of alienation from others and social withdrawal, suspiciousness, and inability to trust others. Although many empiric findings and clinical observations are consistent with this proposal [15,16•], many contend that the lack of specificity of the complex PTSD as well as its overlap with many personality disorder criteria limits its descriptive usefulness [17].

A trauma-related diagnosis recently incorporated into the International Classification of Diseases, edn 10 (ICD-10) is the *enduring personality change after catastrophic experience* (EPCACE). Features include pervasive hostility and mistrust, social withdrawal, feelings of emptiness, hopelessness, being chronically on edge, and estrangement. This diagnosis was considered for inclusion in the DSM-IV [18].

Rather than adding a separate category, the final decision of the DSM-IV Task Force was to list these features under associated features and disorders in the text for PTSD [19]. A more recent study examining expert opinions of EPCACE found considerable endorsement for this new diagnosis, and a recognition that PTSD is too limited in covering the range of possible consequences of human-engendered trauma [17]. However, due to concerns regarding lack of specificity and overlap in features with other disorders, concerns similarly expressed with regard to CP, Beltran and Silove [17] suggest a need to consider probabilistic models with different types of trauma, as certain types of traumas (prolonged, life-threatening, inducing shame or guilt, intentional) are more likely to increase the risk of personality change.

The well-documented unique pattern of increased cortisol suppression among individuals with PTSD provides an interesting forum in which to examine the diagnostic interface of trauma and personality disorders [20]. Grossman *et al.* [21] examined hypothalamic-pituitary-adrenal (HPA) axis alterations among those with personality disorders (BPD vs other PD) with and without PTSD, and found that even after controlling for trauma severity, a significant association between cortisol suppression and PTSD diagnosis remained. Thus, the well-replicated finding of cortisol suppression among subjects with PTSD was found only among those subjects who met diagnostic criteria for PTSD, and not among personality disordered subjects who experienced trauma but did not meet criteria for PTSD.

A recent study examining the association of personality disorders, history of trauma, and PTSD in a sample of anxiety disorder subjects yielded similar findings [22••]. When comparing subjects with no history of trauma, subjects with a history of trauma but no history of PTSD, and subjects with current or past diagnosis of PTSD, those in the latter group were most likely to meet criteria for BPD and self-defeating personality disorder, thereby suggesting that it is the diagnosis of PTSD rather than the experience of trauma that is associated with specific personality disorder features. Therefore, the reviewed recent studies do not suggest the need for an independent diagnosis such as complex PTSD to describe trauma exacerbated personality disorder features in the absence of PTSD.

Despite criticisms that these newly suggested diagnoses lack specificity, they at least highlight the need to address the existence of shared symptoms or features, experienced by both trauma and personality disorder populations. Dissociation, a feature classically associated with trauma and conceptualized as a defense mechanism strategy that has become maladaptive outside of the trauma-evoking situation, is an example of a symptom that is often seen among both trauma survivors as well as those with BPD. However, the presumed etiologic association between trauma and dissociation has recently come into question. A series of empiric studies examining these associations

failed to find any relationship between either sexual or physical abuse and dissociation [23,24]. A recent study among 95 patients with mixed personality disorders found that although BPD diagnosis was significantly associated with pathologic dissociation, there were no significant associations between trauma history among individuals with BPD and pathologic dissociation [25]. In contrast, Zanarini *et al.* [26•] have found that BPD and exposure to trauma, specifically caretaker sexual abuse, are both important risk factors that in conjunction, explain the dissociative experiences reported by BPD patients. The cumulative evidence, some of which challenges long held beliefs, supports the possibility of multiple pathways to dissociation and highlights the complexity of the interface of BPD and trauma.

Parameters of Trauma As Risk Factors for Personality Disorders

It has been estimated that among adults with histories of sexual abuse, 80% have no demonstrable psychopathology [6]. It is also estimated that approximately 40% of BPD patients have not experienced any significant form of trauma [27]. Furthermore, although approximately 34% to 62% of women in the population are exposed to childhood sexual abuse, it is estimated that only 2% of the population meet criteria for BPD [3]. Based on one 30-year longitudinal study, it is estimated that 25% of children exposed to severe trauma develop demonstrable psychopathology as adults [28]. Given these base rate statistics, it is evident that trauma does not necessarily lead to adult psychopathology, and that resilience is quite prominent among trauma survivors.

Examinations of trauma-related risk factors for the development of adult psychopathology have indicated that relationship to perpetrator, sexual acts involving penetration or force, and childhood sexual abuse of greater frequency and duration consistently predict worse outcome. Paris and Zweig-Frank [29] examined whether these parameters, assessed in multivariate analyses, have a specific relationship with the BPD diagnosis. As predicted, there was a higher frequency of multiple perpetrators and sexual abuse involving penetration among the BPD patients when compared with non-BPD psychiatric patients. However, in this study, only 25% of the BPD group experienced penetration or abuse by multiple perpetrators. Therefore, the authors suggest that there may be a subgroup of BPD in which childhood sexual abuse plays a particularly important role, but that it would be premature to suggest that trauma has a specific etiologic role in BPD [29].

Mediation Models

The long-term impact of trauma is likely to depend on interactions with preexisting personality traits and the

presence or absence of protective factors, with the most severe effects being experienced by those who are predisposed to psychopathology [30]. Because most childhood abuse occurs in the context of family dysfunction, it can be difficult to differentiate the various contributions to adult psychopathology. In situations of caretaker abuse, temperament of either child or caretaker would presumably affect the quality of their interactions, including exposure to abuse. Although there is insufficient evidence to suggest that temperament accounts for the development of personality disorders in traumatized individuals, one recent twin study has demonstrated that genetic factors account for approximately 50% of the variance in borderline and avoidant personality disorders [31]. A further complication is the bidirectional influence of trauma, such that exposure to trauma is likely to cause changes in neuroendocrinology, behavior, cognition, and styles of interpersonal interactions that may increase susceptibility towards future exposure to trauma.

The quality of early childhood attachments has been a prominent focus in both trauma and BPD literatures. Laporte and Guttman [32] found that in comparing the childhood experiences of traumatized women with BPD and traumatized women with other personality disorders, those with BPD not only experienced more types of abuse at the hands of multiple perpetrators, but also experienced more losses, than those diagnosed with other personality disorders. Some studies go further in asserting that the quality of maternal attachment outweighs that of abuse in the development of BPD, as evidenced by a stronger association between BPD and impaired family attachments than with presence of sexual abuse [33]. However, recruitment from nonclinical populations and reliance on qualitative data limits the conclusiveness of these latter studies. Regardless, they are compatible with earlier studies suggesting that family dysfunction and lack of parental support may be more predictive of psychiatric problems than childhood sexual abuse [34,35].

A recent empiric study examining the relationship of family environment, childhood sexual abuse, and BPD diagnosis found that diagnosis and sexual abuse predicted different family environments [36]. BPD diagnosis predicted lower scores on cohesion, expressiveness, active-recreational orientation, and intellectual-cultural orientation, and higher scores on conflict, whereas sexual abuse predicted lower scores on organization, moral-religious orientation, and expressiveness [36]. These results seem to suggest that there are multiple pathways to BPD, *ie*, via family dysfunction or sexual abuse. Zanarini *et al.* [37] found that among their sample of BPD subjects, childhood experiences of both abuse and neglect were not only significantly more common than among control subjects, but that these experiences were essentially ubiquitous among the BPD group. Not surprisingly, sexually abused BPD subjects experienced more chaotic environments than their nonabused BPD counterparts. When all factors were

considered together, sexual abuse and neglect by caretakers of both genders were both predictive of the BPD diagnoses [37]. These findings also support a complex etiology for BPD involving multiple pathways that may include childhood sexual abuse and bipolar neglect.

The observed overlap between trauma and BPD has also been attributed to shared dysfunction in multiple neurobiologic systems. The impact of ongoing trauma on the noradrenergic, serotonergic, and HPA systems, is well substantiated. Chronic responses to overgeneralized cues as manifested by individuals with PTSD are generally maladaptive. Studies utilizing the α_2 -antagonist yohimbine have provided direct evidence of noradrenergic dysregulation among individuals with PTSD [38]. More recently, the effect of yohimbine as a probe of noradrenergic activity was compared to the effect of meta-chlorophenylpiperazine (m-CPP) as a probe of serotonergic activity in patients with PTSD [39]. In this study, patients with PTSD and healthy controls were administered yohimbine, m-CPP, or saline on three separate test days in a randomized, double-blind, balanced order. The results indicated that among individuals with PTSD, yohimbine-induced panic attacks tended to occur in different patients than m-CPP induced panic attacks, thereby suggesting that PTSD may involve two neurobiologic subgroups with dysfunctions in either the noradrenergic system or the serotonergic system [39]. Although these findings have tremendous implications for psychopharmacologic interventions for PTSD patients, it should also be cautioned that interrelations between neurotransmitter systems make it difficult to ascertain specific effects with any degree of certainty.

The most compelling evidence of biologic alteration among individuals with PTSD exists in the lower than expected levels of cortisol that is consistently observed among this population. In conditions of stress, there is typically a direct relationship between level of stress and level of cortisol, and an interactive relationship between catecholamines and cortisol, such that cortisol assists in shutting down further sympathetic activation and limits its own production through negative feedback inhibition. The conventional explanation has been that low levels of cortisol among PTSD subjects reflects a chronic adaptation to stress, which in turn results in a chronic inhibition of the HPA axis. However, recent prospective, longitudinal studies of trauma survivors in which cortisol levels were monitored immediately after the trauma and at subsequent intervals, have challenged the assertion that low cortisol levels in PTSD is a byproduct of an adaptation to one-time high elevations of cortisol [40,41•]. In these studies, subjects who developed PTSD had a lower cortisol response immediately after the traumatic event compared with those who did not develop psychiatric disorders, and those who developed major depression without PTSD exhibited a higher cortisol response immediately after the traumatic event than those with no subsequent psychiatric

disorders. These findings are compatible with dexamethasone suppression studies, which have found an increased number of glucocorticoid receptors in those with PTSD but a reduced number of these receptors among those with major depression [42,43].

Studies that assess the neurobiologic underpinnings of BPD have also implicated noradrenergic, serotonergic, and HPA dysfunction, though with much less consistency compared to studies among PTSD populations. Studies of peripheral catecholamines in BPD have revealed a persistent hypercatecholaminergic condition similar to what is observed in PTSD [27,44]. This hyperresponsiveness to the noradrenergic system has been hypothesized to mediate impulsive and aggressive behaviors as well as affective instability through an increased reactivity to the environment [45]. The serotonergic system has been similarly implicated, as indicated by clinical trials of selective serotonin reuptake inhibitors (SSRIs) resulting in the reduction of impulsive and aggressive symptoms [46,47]. Furthermore, m-CPP challenges in BPD subjects have resulted in the presence of a spacey/high behavioral response that is frequently observed among this population as depersonalization or derealization [48]. All of this suggests that many of the symptoms associated with BPD can be attributed to low serotonergic functioning. However, this is far from conclusive given the inconsistent findings on the level of serotonergic functioning in BPD as well as the effectiveness of SSRIs in treating symptoms of BPD.

Studies examining HPA axis alterations in BPD have yielded conflicting reports. As reviewed earlier, Grossman *et al.* [21] examination of HPA function in personality disordered subjects with a history of severe psychological trauma found cortisol hypersuppression only among those who were diagnosed with PTSD. However, an unexpected finding was the low baseline cortisol level observed among BPD subjects, regardless of whether or not they have experienced a DSM-IV criteria A trauma. The baseline cortisol level among their BPD subjects was even lower than levels among PTSD subjects in other studies. This suggests that BPD subjects, regardless of history of trauma, may have some neuroendocrine similarities to PTSD subjects.

Treatment Implications

Numerous empiric studies have given support to various psychotherapeutic treatments for trauma, most of which are centered on exposure based interventions, such as flooding or systematic desensitization in cognitive-behavioral or behavioral treatments. A recent meta-analysis of controlled clinical trials of psychosocial treatments for PTSD of varying sources (*ie*, combat, crime, severe bereavement), found that the impact of psychotherapy on PTSD and other psychiatric symptoms was significant, and that the effect of treatment was maintained

at follow-up intervals [49••]. Exposure based interventions were the most frequently assessed by these studies, though other modalities such as psychodynamic, hypnotherapy, stress inoculation, and supportive counseling were also assessed. The meta-analysis did not find superiority of one therapeutic modality over others.

In contrast, conventional exposure techniques have not been widely used or validated for the treatment of BPD. The treatment that has received the most empirical support in the treatment of BPD is dialectic behavior therapy (DBT). In DBT, treatment progresses through hierarchic stages, with advancement being possible only when targets of lower stages of treatment have been achieved [50]. For example, the first stage of treatment involves the attainment of basic living capacities, which includes the reduction of suicidal behavior as the primary target, forming a collaborative therapeutic relationship as a secondary target, and leading a life of reasonable quality and acquisition of skills as higher-order targets. Contrary to other psychosocial therapies, childhood sexual abuse or other traumas, are not processed in stage 1, but are discussed only to the extent that they interfere with the target behavior of that stage. After the stage 1 targets are achieved, the patient then progresses to stage 2, where trauma and other aspects of invalidating environments may become the focus of treatment. Treatment during this stage may involve exposure-based strategies focused on processing traumatic events and may be similar to other exposure-based trauma treatments as well as psychodynamic trauma treatments [51].

Although there is a general consensus that the treatment of trauma is more effective when it involves some exposure component, many patients have a difficult time tolerating the intense affect associated with these techniques. Recent efforts have focused on integrating aspects of exposure therapy with selected skills from DBT [52]. Although currently untested, such efforts hold considerable promise in increasing therapeutic adherence and effectiveness in the treatment of trauma.

Relative to the extent that medications are utilized, there is a paucity of randomized, placebo-controlled, double-blind studies assessing the efficacy of psychotropic medications on symptoms of BPD or PTSD. Recent efforts have largely focused on SSRIs for treatment of symptoms associated with both these disorders. Two recent multicenter studies involving a total of 395 subjects, compared the effects of sertraline (12 weeks, up to 200 mg/d) with placebo in reducing symptoms of PTSD [53]. Both studies indicated a significantly greater response rate with sertraline, particularly for the avoidance/numbing symptoms typically associated with PTSD, and often observed among BPD patients. Two published controlled trials of fluoxetine have also reported a significant decrease in PTSD symptoms when compared with placebo [54,55]. With regard to the effect of SSRIs on BPD symptoms, one published randomized

double-blind placebo controlled studies has supported the superiority of fluoxetine (12 weeks, 20–60 mg/d) to placebo [56]. However, subjects who had any other Axis I or II comorbid disorders, suicidal behavior, self-mutilation, substance abuse, or current severe aggressive behavior were excluded from the study [56]. Another recent 12 week randomized, double-blind, placebo-controlled study examined the effect of fluoxetine (20–60 mg/d) on impulsive aggression among those with BPD and other cluster B personality disorders [47]. Results indicated significant improvement on measures of aggression and irritability among those in the fluoxetine condition. Current guidelines for the pharmacologic treatment of BPD is to take a symptom specific approach, such that cognitive perceptual symptoms would be targeted by neuroleptics, whereas affect dysregulation and impulsive-behavioral symptoms would be initially targeted by SSRIs [57••]. Although these few studies offer some promise for SSRIs in the treatment of symptoms associated with trauma and BPD, the relative paucity of such studies, the absence of replication studies, and the possibility that studies yielding negative findings have not been published, calls attention to the need for more controlled studies.

Conclusions

The most recent of research efforts in the area of trauma and personality disorders provide preliminary support for specific associations between types of trauma and personality disorder features. Furthermore, recent findings support the need to examine specific parameters within trauma categories and its correlates, which may also contribute to personality dysfunction, as there are likely to be multiple pathways and risk factors in the etiology of BPD. Future research on the effects of trauma correlates (*eg*, quality of caretaker attachments, loss, invalidating environment), would be particularly informative. Recent advances in biologic psychiatry research are encouraging, and should serve to facilitate future investigations aimed at identifying neurobiologic risk factors for symptoms associated with trauma and BPD.

Most research examining the interface between trauma and personality disorders are based on retrospective data collection without corroborating data from other sources. The use of prospective, longitudinal databases would greatly contribute to clarifying our existing knowledge base. Furthermore, given the vast literature that supports an association between trauma and BPD, an integrated treatment approach utilizing empirically supported techniques seems warranted. Despite statistically significant reductions in symptomatology as reported in treatment studies, personality-disordered trauma survivors continue to suffer greatly and are often clinically perceived as treatment resistant.

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